Refine Search

Search Results -

Terms	Documents
L3 and antibod$	130

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L4

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Search History

DATE: Wednesday, November 16, 2005 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L4	L3 and antibod$	130	L4
L3	(L2 or l2) and lysosom$	132	L3
L2	ucp or ucp2	1211	L2
L1	uncoupling adj protein$	824	L1

END OF SEARCH HISTORY

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      S4        5  RD (unique items)
? show files;ds;t/3,k/all
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         (c) 2005 DECHEMA
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         (c) 2005 Thomson Derwent & ISI
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Set	Items	Description
S1	8778	UNCOUPLING PROTEIN?
S2	11360	UCP OR UCP1 OR UCP2
S3	13	(S1 OR S2) (S) LYSOSOM?
S4	5	RD (unique items)

Set	Items	Description
S1	8778	UNCOUPLING PROTEIN?
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S4	5	RD (unique items)

>>>KWIC option is not available in file(s): 399

4/3,K/1 (Item 1 from file: 5)

0013822629 BIOSIS NO.: 200200416140

Differentiation-dependent expression of cathepsin D and importance of *lysosomal* proteolysis in the degradation of *UCP1* in brown adipocytes

AUTHOR: Moazed Banafsheh; Desautels M (Reprint)
AUTHOR ADDRESS: College of Medicine, Department of Physiology, University of Saskatchewan, 107 Wiggins Road, Saskatoon, SK, S7N 5E5, Canada**Canada

JOURNAL: Canadian Journal of Physiology and Pharmacology 80 (6): p515-525 June, 2002 2002
MEDIUM: print
ISSN: 0008-4212
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

Differentiation-dependent expression of cathepsin D and importance of *lysosomal* proteolysis in the degradation of *UCP1* in brown adipocytes

ABSTRACT: The *lysosomal* protease cathepsin D increased markedly in brown adipocytes during differentiation in primary cultures. Differentiated cells had 20 times the amount of immunoreactive cathepsin D found... ...48 h after cells had been exposed to NE for 7 days. In contrast, exposure of the cells to NE for 7 days increased their *UCP1* content by more than twofold, which returned to basal levels within 48 h of withholding NE. The half-life of *UCP1* under basal conditions and in cells chronically exposed to NE was estimated from reductions in (35S)methionine-labelled immunoprecipitable *UCP1* over 72 h. *UCP1* t1/2 under basal conditions was 3.7+-0.4 days, which was similar to the half-lives of labelled mitochondrial translation products (3.6+-0.8 days). The turnover rates of both *UCP1* and mitochondrial translation products were reduced by NE. The turnover rate of *UCP1* in the presence or absence of NE cannot account solely for the rapid loss of *UCP1* from brown adipocytes upon withdrawal of NE. This loss was reduced when cells were incubated with inhibitors of phosphatidylinositol 3-kinases (PI 3-kinase), previously shown to block formation of autophagic vacuoles. Thus, brown adipocytes acquire a large capacity for both uncoupled metabolism and for *lysosomal* proteolysis during differentiation. Withdrawal of NE, as often occurs in vivo from suppression of sympathetic nervous system activity, would not only terminate thermogenesis but also favor formation of autophagic vacuoles to rapidly reduce the cell content of *UCP1*-containing mitochondria.

4/3,K/2 (Item 2 from file: 5)

0009314320 BIOSIS NO.: 199497335605

Temperature and phenylmethylsulfonyl fluoride sensitive loss of uncoupling

protein in isolated brown adipose tissue mitochondrial membranes
AUTHOR: Desautels M; Dulos R A
AUTHOR ADDRESS: Dep. Physiol., Coll. Med., Univ. Saskatchewan, Saskatoon, SK S7N 0W0, Canada**Canada
JOURNAL: Biochemistry and Cell Biology 72 (1-2): p1-7 1994 1994
ISSN: 0829-8211
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: When a membrane suspension prepared from isolated rat brown fat mitochondria was incubated at 37 degree C for 4 h, a loss of uncoupling protein (*UCP*) immunoreactivity was observed on Western blots. Analysis of (3H)GDP-binding characteristics to *UCP* in isolated membranes also showed a significant reduction in B-max without significant effect on K-d. The loss of *UCP* was not due to protease contamination from *lysosomes* or mast cell granules, since loss of *UCP* was still observed when mitochondria were treated with digitonin to lyse *lysosomes* prior to membrane preparation and when mitochondria were isolated from rats injected with compound 48/80 to degranulate mast cells. Furthermore, loss of *UCP* was observed at alkaline pH and was not affected by inhibitors of *lysosomal* enzymes. Loss of *UCP* immunoreactivity was markedly reduced when membranes were incubated at 4 degree C or in the presence of phenylmethylsulfonyl fluoride, but was not influenced by the...

...of GDP. Overall, these results indicate the presence of a serine protease within brown fat mitochondrial membranes that may be involved in the breakdown of *UCP*.

4/3,K/3 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0314847 DBR Accession No.: 2003-15987 PATENT
Promoting nerve cell generation with a neural cell reactive oxygen species activator, useful for diagnosing, preventing and treating disorders such as cancer, Alzheimer's disease, diabetes mellitus and multiple sclerosis - for use in cancer, Alzheimer disease, diabetes mellitus and multiple sclerosis diagnosis, prevention and therapy
AUTHOR: NEWELL ROGERS M K; NEWELL E; CAMLEY R E; TRAUGER R; RICHARD C; CHRISTIANSEN T; CELINSKI Z; VILLOBOS-MENVEY E
PATENT ASSIGNEE: UNIV COLORADO 2003
PATENT NUMBER: WO 200331643 PATENT DATE: 20030417 WPI ACCESSION NO.: 2003-393449 (200337)
PRIORITY APPLIC. NO.: US 329477 APPLIC. DATE: 20011014
NATIONAL APPLIC. NO.: WO 2002US33054 APPLIC. DATE: 20021015
LANGUAGE: English

...ABSTRACT: reduce co-stimulatory molecule expression on cells of the donor organ, and transplanting the donor organ into the recipient subject. WIDER DISCLOSURE - Oligonucleotides, dominant negative *lysosomal* membrane polypeptides, antigens and colloidal dispersion systems used in the methods of the invention, are also disclosed. BIOTECHNOLOGY - Preferred Method: The neural cell ROS activator...
...growth factor) alpha and beta, and lymphotoxin. The ROS activator in the method of (1) is alpha interferon, lipoproteins, fatty acids, cAMP inducing agents, a *UCP* expression vector, a B7.1, B7.2 or CD40 expression vector, angiostatins, angiogenics, viral components, and

exposure to sub-toxic microwaves or low dose radiation...

... product from HIV Nef, HIV tat, and adenoviral E1B. The activator of ROS is an inhibitor of glutathione or glutathione S reductase, superoxide dismutase or *lysosomal* *UCP* and/or is exposure to microwaves. The cell is a nerve cell or a neutrophil. The compound for modulating ROS is an inhibitor of ROS...

4/3,K/4 (Item 2 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0265635 DBR Accession No.: 2001-05389 PATENT
Inhibiting plasma membrane uncoupling protein expression in tumor cells and rapidly dividing bacterial cells, for treating cancer and infectious diseases - uncoupling protein-inhibitor drug screening for chemotherapy sensitization of tumor cell
AUTHOR: Newell M K
CORPORATE SOURCE: Burlington, VT, USA.
PATENT ASSIGNEE: Univ.Vermont 2000
PATENT NUMBER: WO 200078941 PATENT DATE: 20001228 WPI ACCESSION NO.: 2001-102716 (2011)
PRIORITY APPLIC. NO.: US 140574 APPLIC. DATE: 19990623
NATIONAL APPLIC. NO.: WO 2000US17245 APPLIC. DATE: 20000622
LANGUAGE: English

ABSTRACT: Inhibiting plasma membrane uncoupling protein (*UCP*) expression in a cell, comprises contacting a cell with a plasma membrane *UCP* -inhibitor. Also new are: a composition of a plasma membrane targeted *UCP* -inhibitor; sensitizing a resistant tumor cell to a cytotoxic therapy by expressing a functional *UCP* or *UCP* fragment in a plasma membrane of a resistant tumor cell to sensitize the resistant tumor cell to a cytotoxic therapeutic; screening a tumor cell for...
... for screening a tumor cell or a subject for susceptibility to treatment with a chemotherapeutic agent; inducing cellular division in a growth arrested cell; regulating *lysosomal* pH by modifying *lysosomal* *UCP* activity in a cell; treatment of autoimmune disease by administering *UCP* activator; a composition of a *UCP* -inhibitor; therapy or prevention of infection using a *UCP*-inhibitor; nucleic acids encoding *UCP*; and transgenic animals and cells transfected with *UCP*. (106pp)

4/3,K/5 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

138319665 CA: 138(21)319665r PATENT
Reactive oxygen activators or inhibitors for modulating co-stimulatory molecule expression and cell growth as well as treating neuronal, neoplastic, infectious and autoimmune diseases
INVENTOR(AUTHOR): Newell, Rogers Martha Karen; Newell, Evan; Camley, Robert E.; Trauger, Richard; Richard, C., III; Christiansen, Thomas; Celinski, Zbignlew; Villobos-Menvey, Elizabeth
LOCATION: USA
ASSIGNEE: The Regents the University of Colorado
PATENT: PCT International ; WO 200331643 A2 DATE: 20030417
APPLICATION: WO 2002US33054 (20021015) *US PV329280 (20011012) *US PV329477 (20011014)

PAGES: 51 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12Q-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; OM; PH; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG
?